DOWNLOAD PDF THE CLINICAL AND SCIENTIFIC BASIS OF MYALGIC ENCEPHALOMYELITIS-CHRONIC FATIGUE SYNDROME

Chapter 1 : Fading Fast: Women And Chronic Fatigue - Page 10

"The Clinical and Scientific Basis of Myalgic Encephalomyelitis--Chronic Fatigue Syndrome" is an unparalleled resource for patients and clinicians trying to understand this baffling illness. The Nightingale Foundation's ground-breaking definition of M.E. was drafted to quickly diagnose and effectively treat M.E. and also to "give comfort to M.E.

J of Chronic Fatigue Syndrome, Vol. All this work has been pursued by many colleagues in the U. Unlike pesticides and other inorganic substances viruses can replicate and mutate hence there can be epidemic and endemic outbreaks. In other countries other viruses have been involved. However, in our work this has not occurred in epidemic or endemic forms as outlined here. The acute stage may have varying presentations which may be transient and mild, or severe and persistent. These presentations may be respiratory infections or cardiac infections pericarditis, endocarditis or myocarditis resulting in a constrictive pericarditis or a cardiomyopathy. Not only so but Bornholm disease may occur with severe spasmodic pain in the chest simulating angina or abdominal pain simulating appendicitis, cholecystitis or pancreatitis. We should consider the terms applied to the effects of infection in the nervous system due to the poliovirus, which is the enterovirus. Hence the effects were on the "motor" system with some paralysis. Hence, the results are more noticeable to the patient than the doctor, which is not so with poliomyelitis. These pathological results may not be confined to the nervous system but may result in neurohormonal or other differing end organ effects. In CNS cases seen over decades by one of us Dr. From these figures it can be shown, that besides the female preponderance, the total number of varying CNS syndromes are greater for females as can be seen in the table. Moreover, thousands of serological viral tests including IgM, IgG and VP1 performed by us were positive and confirmed this relationship. Certain CNS deformities also resulted in the offspring of mothers who had conceived whilst the serological tests were positive for enterovirus, prior to, or during pregnancy. In the first trimester these included agenesis of the septum lucidum resulting in blindness, agenesis of the corpus callosum, and also amyelination of white matter resulting in severe and diffuse lack of development. In viral infection in the late stages of pregnancy, fully developed organs were affected and we have shown cardiac effects such as endocardial fibroelastosis in two neonates , as well as brain damage in others. These cases have been recorded on MRI, videotape and, sadly, at autopsy. This diffuse variety of initiating and consequential illness is briefly reported here to show that we are not dealing with a simple, single entity, but with serious consequential illnesses. A summary of the CNS syndromes which followed an initial, serotype positive, viral infection is shown in the table next page. The percentage of M. Nowhere is a variety of systemic symptoms seen more often than in this syndrome. Thus much has been claimed by some to be purely psychiatric in origin, with labels ranging from simple depression to hysteria, and other vague hypotheses. See table at http: They may follow an acute viral illness such as Bornholm disease, viral perimyocarditis, labyrinthinitis, or viral meningitis. A vague flu-like illness affecting the chest or bowels may be a harbinger of a more serious consequence. The malaise not only fails to recover, but becomes more defined, developing symptoms such as anosmia or marked concentration difficulties in a previously highly accomplished brain which cannot now recall a paragraph after reading it several times. Initially, muscle power may not appear to be affected but, if examined carefully, tender "softened spots" may be found in calf muscles and sometimes in the recti-abdomini. One such case had to have two operations for hernias which resulted from such "infarctions. We find over the years that the history of the illness written by the patient is of enormous help and we have preserved these histories over four decades and the symptoms which were commonly reported in over of these histories formed the basis of the Score chart which we in the Newcastle Research Group use. A score of 15 on this chart is highly suggestive of M. A full and careful examination, as follows, is mandatory. CHEST This not only involves the lung fields but also careful attention should be given to heart sounds as a pericardial friction rub in some cases can be detected at the lower sternal border. This often signifies a pericarditis. Labile recordings can be found in some cases and, no doubt, account for the reports of "fainting attacks when

standing erect" and a few have fainted see Score Chart. This labile blood pressure is usually considered to be due to neuro-vasomotor instability. In poliomyelitis and other syndromes primarily affecting the efferent pathways, attention to reflexes, etc. Thus the 12 cranial nerves need to be considered first as certain abnormal sensations relating to the cranial nerves are found in these patients. Afferent and efferent responses will be found. See Newcastle Research Group M. There may be saccadic movements which can be recorded and these affect the reading of small print. In some cases there is a reversal of the Argyl-Robertson pupils, often seen in young patients, and this is often not noticed by the examining doctor. It is described in "Diagnosis of Nervous Diseases" by Purves Stewart and occurred in other cases of encephalitis. The pupils are dilated and do not respond to accommodation and also feebly to light. We have videoed these, with a control, often a parent, sitting next to the patient. The video recordings of these cases are retained. Occasionally the trochlear nerve is affected as its nucleus is situated in the midbrain area and this can be assessed by asking the patient to focus in a downward direction, when there is diplopia aggravated by looking sideways at the same time. The effects of light are more noticeable and many patients have to wear very dark glasses. This is NOT due to abnormalities in the receptor areas in the calcarine nuclei, but is due to midbrain "reading" of these visual images by forward projecting fibers from the calcarine nuclei. Eye movements should obviously be carefully assessed in all directions and any nystagmus, diplopia, oscillopsia or other abnormal signs noted. In this connection the excellent work done by Alfredo A. In some of these cases where MRIs have been performed unidentified objects UBOs have been noted and are considered to be due to such "cuffing" in the Virchow Robin spaces which contains CFS between the meningeal coating and cerebral blood vessels. In some cases lack of sensation may be found unilaterally in one discrete area and very occasionally some paresis. The latter has also been videotaped. Some modalities of sound, chiefly high pitches, have very adverse, irritating effects. This is also evident in animals. The audiometer test is very helpful to assess and record the frequency and levels at which intolerance occurs. This involves the elevation of the arms above the head and, if positive, the palms will face outwards due to internal rotation of the whole limb with pronation of the forearm. This is also reported by Purves Stewart and was seen in chorea and other cases of encephalitis. This sign is not specific for M. A case referred recently as M. Nevertheless this simple test should be performed and we have videotaped a number of cases. The color the palmar surfaces of the hands should be noted in the acute stage, as the HFAM disease which occurs in some patients in the primary stages of the illness with conjunctivitis due to enteroviral infection, results in palmar erythematous changes. Subscribe Apart from the usual examination it is wise to do an ultrasound test. A simple fetal monitor can be used. We define this as the "boiling bowel syndrome" as it sounds like this on the sonic scan. He postulated that the trigger factors could be neuroneural or neuroendocrinal, resulting in the change of myoelectric activity from the normal 6 cycles to the faster 3 cycles per minute. Blood flow can be assessed by palpation and ultrasound. The brainstem is "softened" in M. Thus the value of response should be noted initially and subsequently as, mild, moderate or severe. In addition, muscle jitter should be assessed. In some cases rapid jitter can easily be felt, seen and again videotaped. As with the upper limbs the temperature should be noted as in M. It is often much lower than normally would be expected. This balance incoordination is frequently seen to be associated with the upper limb positive, pronator sign. Again it is not confined to M. It presents itself as a rather spastic gait and, in M. E, the patient very often requires personal, or walking-stick support and often a wheelchair. It is not intended to discuss this in detail as the Score Chart and the patients own written histories will make this evident. Suffice to say that it can be verified by discussion. Moreover, if questioned many patients will recall very odd verbal and motor performances, e. As shown by the Score Chart they find it difficult to talk or communicate with friends or relatives for long periods, apart from their sensitivity to sound, mentioned earlier. These various changes of activity are noted frequently by relatives as also the periodic blanching of the face. MRIs in some cases, if there is venous cuffing in the optic fundus may be interesting. Papers on these have been published in the Journal of Chronic Fatigue Syndrome, Thorough serological tests should be done and involve routine blood counts, liver function tests, CPK and an autoimmune profile. Varying hormonal tests should be done if there is

suspicion of end-organ dysfunction. A full viral screen should be mandatory and include the enteroviral groups and IgM and IgG and, if possible the VP1 test. An ESR should be done and repeated as necessary. If raised it would indicate a non-specific inflammatory reaction, which requires further investigation. Tests for thyroid and adrenal function are helpful. In one case we had a patient who, following an illness with high titers to Coxsackie virus developed a mild proptosis, but had no goiter. This patient was seen by a junior hospital doctor and was inadvisably given carbimozole and, later when seen at home, was almost in a myxoedematous coma. Freckles occur on the forehead, face and neck as well as discoloration of the areola, lips, mouth, vagina and other mucous membranes. In this patient and others we have seen there is, if anything, a blanching of the skin and a remarkable lack of pigmentation. To distinguish between failure due to hypothalamic or pituitary stimuli, a test using corticotrophin-releasing hormone CRH is useful. Patients with hypothalamic failure DO respond, whereas those with pituitary failure do not. However, as with any such illness there are obviously feelings of oppression and frustration and a desire to "try anything that would help.

Chapter 2 : What is Chronic Fatigue Syndrome & Myalgic Encephalomyelitis? - Prohealth

Myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS) is a serious, long-term illness that affects many body systems. People with ME/CFS are often not able to do their usual activities. At times, ME/CFS may confine them to bed. People with ME/CFS have severe fatigue and sleep problems. ME/CFS.

Even though the case definitions of the two illnesses do not match, specialists in the US who treat chronic fatigue syndrome acknowledge that the diseases are fundamentally the same. The primary characteristics of the illness are a profound, unrelenting loss of energy that is not relieved by rest; post-exertional malaise, which is a worsening of all symptoms following minimal mental or physical exertion; sleep disorder; cognitive impairment resulting in slowed processing of information, reduced focus and attention, and pain. Because cells of all three systems share the same receptors, any illness that affects one of these systems will affect the other two. The most consistent among these abnormalities is immune system dysfunction, notably reduced natural killer cell NK function. NK cells provide a rapid response to viral infections and tumor cells. Reduced NK function indicates, among other things, that the immune system is unable to clear viruses. One of the earliest immune system studies was performed in by Barker et al. Ojo Amaize et al. The most recent of the NK studies, conducted by Brenu et al. The immune system changes minute by minute. The persistent upregulation of pro-inflammatory cytokines is a marker of chronic inflammation and persistent viral infection in many disease states. More recently, Maes et al. Other immune irregularities, such as antibodies to cardiolipin, seem to indicate that an autoimmune process is involved Hokama. Anticardiolipin antibodies ACA are found in autoimmune diseases such as lupus, rheumatoid arthritis, autoimmune hepatitis, and scleroderma. Any chronic illness points to an immune system that is not operating efficiently. In the early s, the late Dr. The autonomic nervous system regulates homeostasis in the body: Using a similar technique, a group of Dutch researchers in Holland led by de Lange, mapped structural brain structure and volume in CFS patients and healthy controls with high-resolution structural magnetic resonance images using voxel-based morphometry, a form of statistical analysis that measures the shape, size and position of brain structures. Reductions in both white and gray matter have been found by subsequent researchers Barnden, Puri et al. In , Puri et al. They also found that inflammation in certain areas of the brain â€" the cingulate cortex, hippocampus, amygdala, thalamus, midbrain, and pons â€" correlated with the symptoms. Patients who reported impaired cognition, for example, showed neuroinflammation in the amygdala, which is known to be involved in memory. The endocrine system is a collection of glands that secrete hormones directly into the circulatory system. These hormones are then carried to a target organ. The major endocrine glands are the pineal gland, hypothalamus, and pituitary gland in the brain; the pancreas; the sex glands ovaries, testes ; the thyroid and parathyroid glands; the gastrointestinal tract; and the adrenal glands. When glands signal each other in sequence this is referred to as an axis, e. In a research team led by Dr. The study showed decreased levels of cortisol, blunted response of the pituitary gland to corticotropin releasing hormone CRH , and enhanced sensitivity to adrenocorticotropic hormone ACTH. Subsequent studies by Dinan et al. Growth hormone is released during sleep by the pituitary gland. Low GH is associated with a loss of vitality in adults, or, in common parlance, fatigue. Mitochondrial Impairment The mitochondria are small structures within cells that produce adenosine triphosphate ATP , the molecule that generates cellular energy. ATP is essential for every function in the body, which means that when the powerhouses of energy production, the mitochondria, are damaged and levels of ATP decline, the body literally runs out of energy. Melvin Ramsay, the physician who documented the Royal Free Hospital outbreak in the s. In an article written in , Dr. Behan found concrete evidence of damaged mitochondria in muscle biopsies of patients with post-viral syndrome. Out of 50 samples, mitochondrial degeneration was obvious in 40 of the biopsies. Because the heart is composed of muscle tissue, cardiac function will be affected by reduced mitochondrial output. A study by Peckerman et al. Thus, there might be periods in daily activities when demands for blood flow are not adequately met,

compromising metabolic processes in at least some vascular compartments. If cardiac output is low to begin with, standing up would reduce it to levels so low that a person would experience dizziness, black-outs, and even fainting. Once cardiac output is sufficiently reduced, all other systems would decline as well. The body, in an effort to supply blood to the heart, would restrict blood flow to other organ systems, such as the gut. As a consequence, digestion would be impaired, with resultant dysbiosis, malabsorption, and a host of GI problems. Blood flow to the brain would also decline, leading to cognitive problems, depression and anxiety, and impairment of hypothalamic function. With reduced blood flow, the immune system, which depends on the vascular system for transport, could not function efficiently; latent viruses and secondary infections would proliferate. The liver, an organ which relies heavily on blood flow, could not detox the body sufficiently, leading to a buildup of toxins. Under these conditions, exercise would not only strain the system, it could be dangerous. Her explanation of mitochondrial damage is that when the body is stressed, the demands placed on ATP production can exceed the supply. The net result is exhaustion at the metabolic level. In a study conducted in by Dr. Myhill and two colleagues, Dr. However, they could not determine whether the damage to mitochondrial function was a primary effect, or an effect secondary to cellular hypoxia or oxidative stress. Oxidative stress is both the product and the cause of mitochondrial dysfunction. That being said, there is also ample evidence that viruses can directly cause extensive damage to mitochondria. The herpes simplex virus causes massive damage to mitochondrial DNA, contributing to cell death and tissue damage. Viruses can also use mitochondrial proteins for replication, induce cell apoptosis cell death , and increase the production of free radicals. Both illnesses are heterogeneous, manifesting in different organs and at different rates of severity. Both are difficult to diagnose. And finally, exercise intolerance is a hallmark characteristic of both illnesses, a feature which no doubt led Dr. Ramsay to conclude that ME was a mitochondrial disease. The researchers found higher levels of IL-1, TNF alpha, neopterin and elastase an enzyme that destroys bacterial proteins than in controls, indicating an immune response to bacteria in the bloodstream. Furthermore, IL-1, TNF alpha and neopterin were significantly related to fatigue, flu-like malaise, autonomic symptoms, neurocognitive disorders, sadness and irritability. Excess hydrogen sulfide can also inhibit mitochondrial oxygen utilization. Marian Lemle has identified the chemical component. De Meirleir has explained the cause for its increase in the gut, and Dr. Maes has identified the mechanism through which it causes symptoms- translocation. All of these components directly reflect the influence of the microbiome. The microbiome is the community of bacteria, and other micro-organisms that inhabit our bodies. Often, when researchers refer to the microbiome, they are talking about the trillions of bacteria that inhabit our intestines, and that enable us to digest our food, absorb nutrients, and protect us from pathogens. Most promising for patients is the potential therapeutic value of altering the microbiome to treat these illnesses. When Butt et al. The results of the treatment were promising. Of the 60 patients, 42 responded well to the treatment. Most impressive was the fact that after 15 years, 12 of those patients were still symptom free. Statistics In , Reyes et al. The overall prevalence of CFS was per , persons, which meant , people in the U. CFS was more than four times more common among women per , than among men 83 per , Only 16 percent had received a diagnosis and medical treatment for their illness. According to the CDC, there are currently one million people in the U. That number has since grown. In the Netherlands, prevalence may be as high as 3. Sweden has also reported a high prevalence rate of 2. There have been very few systematic epidemiological studies conducted in Africa, but one study suggested that the rates might be higher in Nigeria than in the US. The prevalence of CFS in a community population in Japan was 1. What is Chronic Fatigue Syndrome? Not what you might think. Its purpose is to help demystify this complicated and often misunderstood illness. She is the author of Chronic Fatigue Syndrome: She also writes a blog, Onward Through the Fog, with up-to-date news and information about the illness, as well as the full text of CFS: A Treatment Guide, 1st Edition available in translation.

Chapter 3 : Chronic fatigue syndrome - Wikipedia

The Clinical and Scientific Basis of Myalgic Encephalomyelitis--Chronic Fatigue Syndrome has 9 ratings and 1 review. Jodi said: An excellent resource fro.

General Discussion Summary Myalgic encephalomyelitis ME is an acquired complex disorder characterized by a variety of symptoms and physical findings potentially affecting multiple systems of the body. Many cases are preceded by a viral infection, usually a flu-like or upper respiratory illness, although ME can also be preceded by a non-viral illness or other trauma such as chemical exposure. Onset is usually rapid acute but gradual onsets are also reported. Affected individuals do not recover from the infection and instead experience a wide variety of symptoms including an inability to produce sufficient energy to meet daily demands. Marked fatigue and weakness, sickness, cognitive dysfunction and symptom flare-up follows physical and cognitive exertion. Additional symptoms that may occur include headaches, pain, muscle weakness, neck pain, vision abnormalities such as blurred vision , a sensation of tingling, burning or numbness of the extremities paresthesia , bladder and bowel dysfunction, and sleep dysfunction. Cardiovascular abnormalities have also been reported. Myalgic encephalomyelitis is a chronic and disabling disorder. Severe cases often leave affected individuals bedridden or housebound. Myalgic encephalomyelitis may occur as an outbreak that affects a large group of people epidemically or may only affect an individual non-epidemically. Introduction There is significant controversy and debate in the medical literature about the relationship between myalgic encephalomyelitis and chronic fatigue syndrome CFS. The first outbreak of myalgic encephalomyelitis was in and the term myalgic encephalomyelitis first appeared in the medical literature in Myalgic encephalomyelitis is recognized as a distinct disorder and has been classified as a specific neurological disorder by the World Health Organization WHO since The criteria focused more on fatigue than the encephalitic inflammation of the brain features of the disorder. This was unfortunate, since there is more than sufficient robust evidence which illustrates the underlying biological process involving the central nervous system, immune system, energy metabolism and stress system. Consequently, the emphasis on fatigue unfortunately led to defining the disorder being seen as a psychiatric illness. Because little was known about the cause or physiology of CFS, a wide range of patients were diagnosed with CFS even though they may have had a variety of conditions and experienced different symptoms. CFS eventually evolved into a larger disease designation that overlapped with myalgic encephalomyelitis. Further, some researchers, physicians, and patient advocacy groups have pushed to abandon the illness label of CFS as they argue it is inaccurate and trivializes affected individuals. They want to reclassify these individuals as having myalgic encephalomyelitis. Other researchers, physicians, and many ME patient advocacy groups have argued against this change, noting that myalgic encephalomyelitis should retain a strict definition as a distinct neurological disease that includes measurable abnormal changes in the brain and central nervous system. Individuals who meet the stricter criteria would be diagnosed with myalgic encephalomyelitis. The term CFS should be reserved for individuals who fail to meet the more stringent criteria for myalgic encephalomyelitis and in whom no other underlying disorder or condition can be identified. Many patients who have been diagnosed with CFS would meet the more stringent criteria for myalgic encephalomyelitis. Individuals who fail to meet the criteria should be retested for an underlying condition as many individuals initially diagnosed with CFS are eventually diagnosed with an underlying condition such as cancer, multiple sclerosis, lupus, brucellosis, or another condition. Three of the more common case definitions include the Fukuda et al. It also requires the presence of at least four of eight symptoms including: Research has indicated that individuals with a primary psychiatric illness e. Lastly, the Myalgic Encephalomyelitis â€" International Consensus Criteria ME-ICC advocates for removing fatigue as a characteristic symptom and defines the disorder as an acquired neurological disease with complex global dysfunctions. The Nightingale Research Foundation, a Canadian charity dedicated to myalgic encephalomyelitis, uses a strict definition that states myalgic encephalomyelitis is an acute onset biphasic

epidemic or endemic infectious disease process, where there is always a measureable and persistent diffuse vascular injury of the central nervous system in both the acute and chronic phases. For more information on specific case definitions, see the Resources and References sections of this report. Unfortunately there is no consensus on nomenclature or classification for these disorders, and different countries, organizations, and researchers continue to use different names to describe these conditions. Until a global consensus is reached on how to name and classify these disorders, confusion will persist. Again, because of the lack of a clear, agreed upon definition of the disorder, different medical sources list different symptoms as being associated with ME. Most sources describe ME as a distinct neurological disorder that can affect multiple systems of the body. Symptoms and their severity can fluctuate over the course of the illness, even from hour to hour. The symptoms discussed below have been associated with different case definitions of ME. It is important to note that ME is highly variable and can affect individuals differently in regard to severity, progression, and specific symptom development. Most individuals will not have all of the symptoms discussed below. Some cases of ME may develop in two phases biphasic. The first phase is an acute primary infection phase. Affected individuals may have an infectious disease with an incubation period of approximately four to seven days. Other cases may follow a more gradual onset. Closely following this initial phase is a second phase known as the chronic phase. This second phase usually occurs two to seven days after the initial infection and is characterized by measurable widespread diffuse changes in the central nervous system, which are thought to be the result of an infection invading the nervous system encephalitis , or the immune system attacking the brain autoimmune encephalitis. Brain inflammation is encephalitis, so ME does appear to be a form of encephalitis. A characteristic or hallmark symptom of ME is marked fatigue, sickness, and symptom flare-up that follows physical and cognitive exertion known as post-exertional neuroimmune exhaustion or post-exertional malaise. Normal activities of daily living can cause severe physical or cognitive fatigue which can last for days, weeks, or even months. A 24 hour delay in the onset of post-exertion is fairly common. Affected individuals develop a lack of stamina that causes a considerable reduction in activity level. Even mild exertion through normal, daily activities is typically associated with worsening of other symptoms. It is important to not underestimate the neurocognitive problems in this disease, in both practical and emotional terms. Significant changes in personality can be present, and will vary according to the underlying cause, the severity of the inflammation, and delays in treatment. Affected individuals may also have a variety of neurocognitive impairments such as difficulty processing information e. For many people, these deficits affect nearly all spheres of their daily activity, severely impacting overall daily functioning levels and posing a significant burden to families and caretakers. A variety of pain symptoms can be associated with ME, including chronic headaches and significant muscle pain myalgia. Additional neurological symptoms may include an inability to focus vision, impaired depth perception, loss of proprioception, visual-spatial disorientation, sensitivity to sunlight, muscle weakness, unsteadiness, and poor coordination. Additional symptoms that can occur in individuals with ME include abnormalities of the immune, gastrointestinal, and genitourinary systems. General, nonspecific symptoms normally associated with the flu or a similar illness may occur. Such symptoms include sore throat, inflammation of the sinuses sinusitis , and abnormal enlargement of lymph nodes. Affected individuals may be particularly susceptible to viral infections. Gastrointestinal symptoms may include abdominal pain, bloating, nausea, and irritable bowel syndrome. Genitourinary symptoms include increased frequency or urgency to urinate and increased urination at night nocturia. Individuals with ME are at risk for cardiovascular symptoms. Affected individuals may experience palpitations with or without irregular heartbeats arrhythmias , low blood pressure neurally mediated hypotension , and postural orthostatic tachycardia syndrome POTS. POTS is a condition characterized by an abnormal increase in the heart rate upon standing. Affected individuals may faint or become dizzy upon standing. Labored breathing, headaches, shakiness, nausea, and fatigue of chest wall muscles may also occur. Affected individuals may also experience abnormalities in the regulation of body temperature, including sweating episodes, cold extremities, and feeling feverish with or without a low grade fever. Some individuals

may be intolerant of extreme temperatures. Additional symptoms that have been reported in ME include seizures, paralysis, and abnormal sensitivity to certain foods, medications, odors, or chemicals. Affected individuals are more likely to have other illnesses or conditions that occur along with ME concurrent or comorbid illness. In children, ME may be characterized by brief episodes of excessive restlessness and movement hyperactivity followed by extreme weakness. Children may rest frequently, which can be misinterpreted as laziness. Mood swings and irritability are common. Children may not recognize symptoms of ME and may not complain. Additional symptoms that occur in children include pain, headaches, memory abnormalities, difficulty processing information, and a decline in school performance. The onset of ME in children is usually around age 12, but has been reported in children as young as 2 years of age. A specific pediatric case definition has been proposed by Jason and colleagues Causes The exact cause of myalgic encephalomyelitis ME is not fully understood, although there are several theories. Most investigators agree that the disorder is most likely the result of an abnormal immune system and brain function in response to an infection or virus. Although the brain and immune system are most likely impaired or abnormal dysregulated in this disorder, the exact underlying problems are unknown. A variety of additional factors that have been theorized as playing a role in the development of ME include genetic and environmental factors. Some studies have shown that ME occurs in greater frequency among relatives to the third degree genetic predisposition. In contrast, some believe that environmental factors play a greater role than genetic ones. However, definitive evidence linking specific environmental factors to the development of myalgic encephalomyelitis is lacking. Some researchers believe that an enterovirus infection could be an underlying cause of the disorder. Enteroviruses are small, contagious viruses consisting of ribonucleic acid and proteins. They are the second most common viral agents in humans, behind only rhinoviruses the viruses that cause the common cold. Enteroviruses can affect anyone of any age. Individual susceptibility to enteroviruses varies. The reason why some individuals develop ME after infection with an enterovirus is unknown. It is not known whether these viruses caused ME or whether they developed due to an impaired immune system in affected individuals. It was once thought that the ability of the blood-brain barrier â€” the specialized capillaries that prevent blood contaminants from entering the brainâ€”to block viral entry into the CNS was adequate; however, more recent evidence indicates entry through other routes, such as along the auditory nerve. Additionally, no one virus has been identified that explains all cases of ME. Some studies suggest that in individuals with ME the viruses can trigger cascading events in the central nervous system through chronic activation of the immune system which, in turn, can result in widespread diffuse neurological dysfunction, changes at the cellular level, and nerve cell injury and death. Even when not actively replicating, an infection can lead to profound dysregulation of the immune response, causing neuroinflammation which destabilizes overall brain function, and producing symptoms with widely fluctuating severity levels Viruses also do not continually replicate, but do so at times of immune vulnerability, such as at times of physical or psychological stress. Unfortunately, viruses go latent, then they reactivate, and repeat this patterns, and once in your cells, any elevation of cortisol levels can cause the reactivation. Affected Populations Because of the controversy regarding the definition and classification of myalgic encephalomyelitis ME and related disorders, determining their true frequency in the general population is difficult.

Chapter 4 : Myalgic Encephalomyelitis/Chronic Fatigue Syndrome: Guidelines for Doctors - Prohealth

Myalgic encephalomyelitis (ME), chronic fatigue syndrome (CFS), and chronic fatigue (CF) are distinguished accurately: results of supervised learning techniques applied on clinical and inflammatory data.

This book provides, in one superb chapter source, an up-to-date, comprehensive account of current knowledge concerning the history, epidemiology, children with M. This is an essential reference book for medical, government and public library reference rooms. This text is a unique vehicle for researchers, physicians and other health education and government officials, and is easily understandable by the general public interested in M. All funds from this text will be used both to promote research and to assist in greater understanding of M. Customer Book Reviews An essential M. This book is the one and only M. This book is essential reading for anyone with M. I cannot recommend them highly enough. As Dr Hyde explains, M. The following are my original recommendation. I strongly disagreed with Dr Caruthers in the merging the definitions of M. It is increasingly obvious that too much importance was being placed upon the definitions of Chronic Fatigue Syndrome CFS , and not enough upon the actual disease, Myalgic Encephalomyelitis M. These two illness spectrums are not the same and should not be considered to be the same. Such is not true of M. One has to cease believing those who patent virus and infectious agents for profit are necessarily going to tell the truth. If one looks only at M. There are no known patents on this group of viruses. Still worth reading after all these years Nearly a decade later, much of the information in this book is still germane - and interesting, such as the historical recounting of epidemics through the years, brain imaging patterns, neurocognitive abnormalities and more. The clinical and scientific basis of Myalgic Encephalitis By Daniel on Apr 22, Unbelievably insightfull to those who have or are suffering from this devestating disease process. Every treating physician should have this in there office. In my personal observations with some people I have met and also myself and my wife it is definetly and as a matter of medical fact a potential lifesaver. The fact that this author Dr. Byron Hyde has went to extreme lenghts to study research and organise the world symposium on this to gather varied medical research and facts from all these brilliant minds tells me that this man deserves at the very least a medal of Honour but rather the Nobel Peace Prize for devoting at least 25 years of his life to studying and caring enough to try and find the cause and cure of so much human suffering that exists in our world. I passed on the book to another fellow sufferer and never really got the chance to read very much but my doctor has one now. A vital resource for patients and doctors! By Boo Radley on Sep 22, When you or someone you love is suddenly stricken with a serious illness, you want an experienced, knowledgeable doctor to be able to diagnose and treat you efficiently and with sympathy. The only way to avoid getting lost in the healthcare system or, even worse, to find yourself participating in prescribed treatment programs that may do more harm than good, is to arm yourself with as much knowledge as you can. The Nightingale Foundation is one of the few bodies dedicated to researching M. It is also one of the few organisations to advocate the distinction between Myalgic Encephalomyelitis and what is commonly called Chronic Fatigue Syndrome - a crucial qualification. I was dumbfounded that so much work and intense effort has been put in to the study and depth that was shown in this work. It is very readable and well organized. The extensive index also helped with the specific information in question. This should be used in ALL medical schools for neurology. I did learn a little, but it was mostly "over my head" as it is written for the medical professional, it seems to me. It confirmed what I had learned online. Excellent information from Dr. Hyde in this book is essential to understanding what ME is. This should be a book each and every person with the above illnesses owns. An Essential for Doctors and patients alike. Obviously no-one can read all this information from cover to cover, but for reference and to dip in and out of for information it is excellent. Add a Book Review Book Summary: Goldstein , Byron M. This particular edition is in a Hardcover format. It was published by Nightingale Research Fndtn and has a total of pages in the book. To buy this book at the lowest price, Click Here.

Chapter 5 : The Clinical and Scientific Basis of Myalgic Encephalomyelitis/Chronic Fatigue Syndrome - ME

Genre/Form: Congresses Conference papers and proceedings: Additional Physical Format: Online version: Nightingale Research Foundation review of the clinical and scientific basis of myalgic encephalomyelitis/chronic fatigue syndrome.

References Immunological aberrations inflammation, immune activation, immunosuppression and immune dysfunction ; Klimas et al. Aberrations at the cellular level include mitochondrial dysfunction Booth et al. Tissue anomalies encompass significantly low oxygen uptake by muscle cells Vermeulen and Vermeulen van Eck, , increased intramuscular acidosis after maximal voluntary contraction with significantly prolonged pH recovery times Jones et al. The distinction between patients meeting the proposed criteria for ME Carruthers et al. Exercise and orthostatic stress seems to induce or intensify long lasting abnormalities in patient subgroups, e. Although the exact etiological mechanisms remains to be elucidated, the post-exertional malaise phenomenon could arguably be explained by the observation that physical stress, particularly anaerobic exercise, can intensify pre-existing abnormalities: Potentially relevant clinical subdivisions are summarized in Table S1 see Supplementary Material. The most relevant subdivisions are based upon exercise-induced immunological abnormalities. Acknowledging immunological, infectious and endocrine subdivisions seem to be crucial to establish the efficacy and safety of pharmacological and behavioral therapies. CFS criteria focus primarily on chronic fatigue, which, due to is nature, is a subjective and ambiguous criterion Jason et al. The question whether the label ME is appropriate Baraniuk et al. Trials into the efficacy and safety of pharmacological Fluge et al. To unravel the enigma and to resolve the controversy, patients should be monitored before, during and after potentially effective therapies in research and clinical practice using objective measures Kindlon, , e. Conclusion Looking at the definitional criteria of ME, e. The introduction of the label CFS, but more importantly a fatigue-based case definition, have resulted into research of a heterogeneous patient population of patients with chronic fatigue. Combined with the use of subjective measures this has created confusion and controversy. Several authors have questioned the validity and nature of symptoms reported by patients. This debate can be resolved by assessing characteristic symptoms using objective methods, if possible, e. Despite the ambiguous CFS criteria and methods applied, researchers have observed various specific abnormalities repeatedly, e. Conflict of interest statement The author declares that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Chapter 6 : Outbreaks â€“ American ME and CFS Society

Chronic Fatigue Syndrome & ME is a "multi-system disease," that is, it affects several systems in your body simultaneously: the immune system, the nervous system, and the endocrine system.

Although it was published more than 20 years ago, it contains a wealth of information that is still relevant today. This list spans the years There have reputedly been outbreaks since in the Middle East, Eastern Europe, and Asia, but documentation is lacking. It is important to keep in mind that many diseases occur in both epidemic and sporadic form. Polio was sporadic for many years before it became epidemic. Polio is now, once again, sporadic. Epidemic among personnel at L. County Hospital, Ruth Protection Home and throughout California, paralleling poliomyelitis, often diagnosed as atypical poliomyelitis. Outbreak of a disease resembling poliomyelitis. Outbreak described as epidemic neuromyasthenia. Chestnut Lodge Hospital student nurses described with poliomyelitis-like epidemic neuromyasthenia. Outbreak among medical and nursing staff in a Liverpool Hospital. Outbreak of benign myalgic encephalomyelitis among Royal Free Hospital staff. Outbreak of Benign encephalomyelitis. An outbreak of encephalomyelitis. Unusual response to poliomyelitis vaccination. Outbreak of acute infective encephalomyelitis simulating poliomyelitis among a residential home for nurses. An epidemic of neuromyasthenia. An outbreak of epidemic neuromyasthenia. Began after an ill serviceman returned home from England. Outbreak described as epidemic malaise and benign myalgic encephalomyelitis. Reports of sporadic cases of myalgic encephalomyelitis. Outbreak of benign myalgic encephalomylitis. Reports of sporadic cases of influenza-like illness Basel, Switzerland: Outbreak described as epidemic neuromyasthenia in a convent in New York State. Outbreak described as epidemic malaise and epidemic neuromyasthenia. Sporadic cases resembling benign myalgic encephalomyelitis. Report on an epidemic of benign myalgic encephalomyelitis. Reports on Mylagic Encephalomyelits and epidemic neuromyasthenia in this region. Coxsackie B outbreak reported in a general practice. Sporadic cases of M. Included here are outbreaks in Dunedin and Hamilton New Zealand. After an apparent initial increase in the morbidity in there seemed to have appeared in late summer of an unprecedented increase of sporadic and epidemic cases across North America. Although certain geographical hot spots seen to have taken up much of the medical interest, this endemic situation probably represents an unusual and unremitting morbidity in all areas of the United States and Canada. Many of these patients were associated in some way with Columbia Community College. Rosedale Hospital reported 11 cases of M.

Chapter 7 : Myalgic Encephalomyelitis - NORD (National Organization for Rare Disorders)

The clinical and scientific basis of myalgic encephalomyelitis/chronic fatigue syndrome. Ottawa: The Nightingale Research Foundation ; [Google Scholar]].

Final requirement All other known causes of chronic fatigue must have been ruled out, specifically clinical depression, side effects of medication, eating disorders and substance abuse. The clinical evaluation should include: A thorough history that covers medical and psychosocial circumstances at the onset of fatigue; depression or other psychiatric disorders; episodes of medically unexplained symptoms; alcohol or other substance abuse; and current use of prescription and over-the-counter medications and food supplements; A mental status examination to identify abnormalities in mood, intellectual function, memory, and personality. Particular attention should be directed toward current symptoms of depression or anxiety, self-destructive thoughts, and observable signs such as psychomotor retardation. Evidence of a psychiatric or neurologic disorder requires that an appropriate psychiatric, psychological, or neurologic evaluation be done; A thorough physical examination; A minimum battery of laboratory screening tests, including complete blood count with leukocyte differential; erythrocyte sedimentation rate; serum levels of alanine aminotransferase, total protein, albumin, globulin, alkaline phosphatase, calcium, phosphorus, glucose, blood urea nitrogen, electrolytes, and creatinine; determination of thyroid-stimulating hormone; and urinalysis. Other diagnostic tests have no recognized value unless indicated on an individual basis to confirm or exclude a differential diagnosis, such as multiple sclerosis. CDC criteria[edit] The initial chronic fatigue syndrome definition was published in Oxford criteria[edit] The Oxford criteria was published in [5] and include both CFS of unknown etiology and a subtype of CFS called post-infectious fatigue syndrome PIFS , which "either follows an infection or is associated with a current infection. The definition is also referred to as the Canadian consensus criteria. Functional impairment must be below defined thresholds in two of the three designated subscales of the Short Form 36 Health Survey i. Vitality, Social Functioning, and Role-Physical. These revised criteria require exercise-induced fatigue; memory and concentration impairment, normally accompanied by other neurological or psychological symptoms; and variability of symptoms, often brought on by mental or physical activity. Based on the "Canadian" definition by Carruthers et al. The main symptom is " post-exertional neuroimmune exhaustion " PENE i. The diagnosis should be reconsidered if none of the following symptoms remain: It expects a diagnosis in a child to be made by a pediatrician. The CDC criteria states diagnostic tests should be directed to confirm or exclude other causes for fatigue and other symptoms. Further tests may be individually necessary to identify underlying or contributing conditions that require treatment. The use of tests for diagnosing chronic fatigue syndrome should only be done in the context of protocol-based research. The following routine tests are recommended: